NARCOLEPSY

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THE recently awakened interest in this uncommon disease has prompted the reporting of a rather typical case.

REPORT OF CASE

J. D., age 30, white male, was first seen January 8, 1935, complaining of an intense and uncontrollable desire to sleep, coming on during the day.

These attacks, noticed first at about eighteen or nineteen years of age, have gradually become more intense and more frequent; and during the year previous to the first examination the attacks were so severe that at times it was necessary for him to stop driving his car, or ploughing, or to cease from other activities at periods throughout the day and take a short nap. He was also forced to give up evening social engagements because, unless there was excitement, he would soon fall asleep. The attacks come on at any time during the day, but are more likely to occur shortly after heavy noon meals, or during warm weather, or after heavy labor, or during uninteresting, quiet situations or prosy conversations.

Each attack of narcolepsy consists of a gradually increasing desire to sleep, which he cannot fight off and which culminates in what appears to resemble in all respects normal sleep. Following from five minutes to an hour or more of sleep, the young man awakens in a "sleepy" condition, but is able to go about his business, rapidly regaining his alertness. When disturbed he feels as though aroused from a normal sleep of inadequate duration, and is always cross and annoyed. He does not seem to have any disturbance of nocturnal slumber, sleeping deeply and usually from eight to nine hours. Loss of nocturnal sleep apparently increases the tendency to diurnal sleep. The patient also complained of occasional spells of weakness, but suggested no connection between these and the attacks of sleepiness. These spells of weakness (cataplexy) manifest themselves in two ways. Both are precipitated by some intense emotional response such as laughter, excitement, or anger. The first consists of an absolute inability to move. For instance, after seeing a very funny moving picture he was absolutely unable to move a muscle of his body. On this and similar occasions he has been shaken by a friend who, used to having him fall asleep, has thought him asleep. This stimulus is sufficient to recall his muscle power instantaneously. He describes the feeling as being "weak as a rag for a minute."

The other manifestation of cataplexy consists of sudden transient spells of weakness, so brief that he has never fallen. These occur during activity of some sort, such as walking, and are brought on usually by excitement, such as seeing a child falling off a fence. In this particular instance (typical of others), with his first step, on running to catch her, "his knees failed him and he almost fell, but he caught himself on the next step." He is unable to recall the age when these attacks first appeared.

The family history is negative, except that his father has always fallen asleep very readily, following meals as well as nocturnally.

He was born and has always resided in California, is actively engaged in farming, has been graduated from a university where he made a good scholastic record and showed a normal, social reaction. In childhood he had measles, mumps, whooping-cough, and chicken-pox. In 1918, at the age of fourteen, he had influenza, which apparently had no relation to the attacks of narcolepsy. He denies any venereal infection, and his blood Wassermann reaction was negative in 1925.

In 1927 and 1928 he was in automobile accidents, but in neither case was he responsible for the accident. He received head injuries, and was unconscious for a few minutes on both occasions; in the first accident there was bleeding from the right ear, and a 10 per cent reduction in hearing has resulted. These injuries did not seem to alter the course or severity of the symptoms of narcolepsy in any way.

Physical examination revealed a well-developed and nourished white male of thirty years, six feet in height, weighing 178 pounds. There were no pathologic findings, except that tendon reflexes could be obtained only on reinforcement.

The laboratory examinations showed a normal urine, hemoglobin 17 grams, and red blood cell count of 5,580,000. The white blood and differential counts were normal. The sedimentation time was over three hours, and the Kahn blood test was negative. The basal metabolic rate was plus 16.

On the basis of this typical history and negative findings, a diagnosis of narcolepsy with cataplexy was made and, following the suggestion of Doyle and Daniels,[1] the patient was given ephedrin sulphate, grain three-eighths, at 7 a. m., noon, and 4 p. m. He stated that he obtained a prompt relief of his symptoms, but that he felt nervous after the four o'clock dose; so this was discontinued after one week. For the next month he took the ephedrin regularly and felt greatly relieved, and occasionally he enjoyed an afternoon nap, but was free of the uncontrollable desire to sleep.

He was not seen again for two and one-half months, during which time he took the ephedrin sporadically. He has been able to fight off attacks of sleep while engaged in any normal activity, but if he lies down he promptly falls asleep.

Following this treatment, he took no ephedrin for one month. There has been no relapse, and he has not had to stop driving his car since first starting ephedrin. He has been able to resume evening social engagements, with the exception of bridge, "which bores him so that he cannot stay awake."

In spite of the improvement in the narcoleptic attacks, there seems to have been no change in the cataplectic phenomena.

COMMENT

This patient presents in typical form the two major manifestations of narcolepsy, the predominating symptom being the occurrence of diurnal naps as a response to an overwhelming desire to sleep, occurring after adequate sleep and rest, several times during the day. The other symptom is cataplexy, which consists of spells of weakness involving a part or the whole of the body. Frequently, as in this patient, the person does not make any connection between the two complaints, and the onset of one may precede the other by a period of months to years.

The etiology of narcolepsy is undetermined, and this patient is typical of many in the absence of any extraordinary findings. Often an interesting family history of abnormal drowsiness or actual attacks of narcolepsy has been obtained. The father of this patient shows abnormal drowsiness.

Of the other diseases showing abnormal drowsiness, syphilis, brain tumor, cerebral arteriosclerosis, hypopituitarism, diabetes mellitus, obesity, polycythemia, malnutrition, and others, this patient evidences none, except a slight increase in the blood count, and this is not of sufficient magnitude to be grouped in the class of polycythemia.

A certain number of cases of narcolepsy have followed encephalitis, and this has led some authors to consider the two conditions as related. This patient had influenza in 1918 and from the history the attack seemed typical. There is no evidence that this might have been encephalitis, and following this attack of influenza there was a period of four to five years before the symptoms of narcolepsy appeared.

1 Doyle, J. B., and Daniels, L. D.: Symptomatic Treatment for Narcolepsy, J. A. M. A., 96:1370-1372 (April 25), 1935.

Patients have responded to a variety of treatments: withdrawal of spinal fluid, intrathecal introduction of air, irradiation of the hypothalamic region, psychoanalysis, and suggestion. Prior to the introduction of ephedrin sulphate, symptomatic treatment was neither satisfactory nor consistent. This drug has produced the most satisfactory results in treatment yet obtained. Some obtain prompt and apparently lasting relief, others slight or transient; but most are apparently pleased with the results obtained.

Usually the response to ephedrin affects the cataplectic tendency more than the drowsiness, but in the patient here reported the effect was just the opposite. What part suggestion has to play in this and responses to other types of treatment is hard to evaluate. However, suggestion is certainly a potent influence on these patients, and the failure of the cataplectic phenomena to respond may be due to the fact that a successful attempt was made to avoid the suggestion that the cataplexy and drowsiness were related.

SUMMARY

A typical case of narcolepsy is presented showing both drowsiness and cataplexy. The patient made a satisfactory response to treatment with ephedrin sulphate.

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AMEBIASIS: ITS DIAGNOSIS BY THE GENERAL PRACTITIONER

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IN view of the fact that from five to ten per cent of the population in California have amebiasis, it is imperative that the general practitioner be qualified to diagnose and treat this disease.

The impression has been created that such diagnosis and treatment is a highly technical and difficult procedure, which can be accomplished by only a few men in the metropolitan areas. This assumption is not warranted by the facts. Amebiasis should be suspected in the patient who presents a number of vague symptoms, usually involving the gastro-intestinal and nervous systems, and apparently without organic basis.

REPORT OF CASE

The following case history of Mr. Mc., age forty-seven, is illustrative: Six years ago he had so-called "intestinal flu," with diarrhea and abdominal distress. Since that time he has not felt very well, but has been able to work. His complaints when first observed by me, April 27, 1935, were: "Gas in abdomen, uneasiness or turmoil of intestines, sensitive belly, incomplete satisfaction following bowel movement, two mucus stools daily, rectal itching, dizziness, palpitation, sweating of palms of hands, nervousness, irritability, inability to eat beans, sweet potatoes, melons, and spinach, because they cause abdominal discomfort and gas."

Diagnosis.—The diagnosis of amebiasis can only be made by the identification of E. histolytica in the stools. The detailed description of the appearance of this parasite and its differentiation from other amebae inhabiting the intestinal tract of man, may be learned from any good book on clinical laboratory methods.[1] An excellent chart for this purpose may be found in an article by Gradwohl in the *Journal of the American Medical Association,* Vol. 102, No. 1, page 66. The cystic forms are more characteristic and more easily differentiated from the other amebae, as they have only four nuclei, as contrasted with eight in E. coli.

In the fresh specimen the protoplasm of the vegetative and of the cystic forms is seen under the low power of the microscope as highly refractile, glistening, greenish-white, transparent, ovoid or spherical bodies. The cystic form is usually about twice (15 microns) the size of a red blood cell, and the vegetative form usually two to three times (20 to 40 microns) larger than a neutrophilic leukocyte. If one is fortunate, he may see the vegetative form actively pushing out pseudopodia or containing red blood corpuscles. Morphology must be studied by the high power or oil immersion lens. A smear of the specimen may be made on a microscopic slide, fixed in Schaudinn's fluid and stained with iron hemotoxylin. This method is certainly better for prolonged study and for the differential diagnosis of the various amebae.[2] Practically, it is a long procedure and not readily done by the general practitioner unless he just fixes the slide in the Schaudinn's fluid and sends it to a laboratory for diagnosis. Here let me sound a warning—not all laboratory workers can identify E. histolytica.

The examination of the fresh stool is the method which I prefer, because it can be done by every physician and, with sufficient practice, it is quite reliable. No bismuth or mineral oil should be given previous to the examination.

Procedure in Obtaining Specimen.—To obtain the proper specimen is most important. I usually examine the first stool without any preparation, having the patient pass the stool into a paper cup at the office. One should choose the soft part of the stool and get any mucus present. One or two loopsful from different parts of the stool, is mixed with a drop of warm physiologic saline on a warmed slide. The following morning the patient is given a drastic saline purge, not allowed to eat breakfast, and told to report to the office after passing the third stool. By this time most of the food is absent from the stool, and one may get mucus. Let me *emphasize* that in the mucus can be found the typical cysts. Sometimes the patient may pass just enough mucus to soil the toilet paper, but one can get an excellent specimen from it. This mucus is placed on a slide with the loop and, if too thick, a drop of physiologic saline is added; then it is covered with a cover glass and searched for refractile protoplasm as described above.

The diagnosis is made by finding the specific protozoa. I never do less than three stool examinations, and with this method more are seldom necessary to find the organism, if it is present.

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1 Todd and Sanford: Clinical Diagnosis by Laboratory Methods, p. 454, Saunders.

2 Magath, Thomas B.: The Laboratory Diagnosis of Amebiasis, J. A. M. A., Vol. 103, No. 16, p. 1218.